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Use Of Neural Oscillators Triggered By Loading And Hip Angles To Study The Activation Patterns At The Ankle During Walking In Humans

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Use Of Neural Oscillators Triggered By Loading And Hip Angles To Study The Activation Patterns At The Ankle During Walking In Humans

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Contents

Summary

Background

Spinal pattern generators (SPG), which are neural networks without a central input from the brain may be responsible for controlling locomotion. In this study, we used neural oscillators to examine the rhythmic patterns generated at the ankle during walking. In addition, we wanted to determine if SPG can adapt to the changing motor demands from a loss of loading afferents, walking at different speeds, and performing silly walks.

Methods

The methods consist of three parts; first, we fit the outputs from the SPG model to experimental electromyography (EMG) data during normal walking. In the first part, seven healthy male subjects were requested to perform silly walks (3.8±0.4 km/h), normal walking at self-selected speed (4.8±0.5 km/h), 3.5 km/h, 4.0 km/h and 4.5 km/h on a treadmill. Force measurements acquired from pressure insoles, EMG and kinematic data were captured simultaneously. The SPG model consisted of a simple oscillator made up of two neurons; one neuron will activate an ankle extensor and the other will activate an ankle flexor. The outputs of the oscillator represented the muscle activation of each muscle. Insole forces and hip angles of six consecutive strides captured from the experiments were used as inputs to the model. A nonlinear least squares algorithm was used to determine a set of parameters that would optimise the differences between model output and experimental EMG data. In the second part, we introduced a perturbation by a sudden removal of the loading input while retaining hip angles in the SPG model, and calculated simulated EMG responses. Thirdly, the model responses were compared with experimental EMG data collected during sudden unloading in four healthy male subjects when they walked across a platform which accelerated downwards by 4 cm in random trials.

Results

Our results showed that it is possible to reproduce muscle activations using neural oscillators. No significant differences in the model parameters were found between normal walking at self-selected speed, and other walking speeds. Only the adaptation time constant for the tibialis anterior during silly walks was significantly different compared to the other normal walking trials. Our model showed co-activation of antagonistic muscles when sudden unloading occurs

during stance. Similarly, our experimental results on four healthy subjects also indicate that activation of the soleus could be mediated by the spinal cord in the absence of an expected loading afferent. However, this was only seen in the perturbed trials during midstance for the tibialis anterior.

Conclusion

A close correlation between simulated and measured muscle activations during normal walking at self-selected speed indicated that spinal control should not be underestimated in models of human locomotion. This also shows that SPG may provide a direct link between sensory inputs and muscle activations via motoneurons. We also showed that SPG in the spinal cord can interpret and respond accordingly to velocity-dependent afferent information. Changes in walking speed do not require a different motor control mechanism so long as posture is not disturbed, and there is no disruption to the alternating muscular activations generated at the ankle. In addition, we found that co-activation during sudden loss in loading afferents might be the first defensive response to sudden changes in a support surface to increase joint stability. For the soleus, simulated EMG data from our model was similar to experimental perturbed trials. However in the tibialis anterior, this was only found during midstance. So, less than expected afferents to the spinal cord might result in mediation by other sources for the tibialis anterior. Furthermore, only the adaptation time constant for the tibialis anterior was significantly different while performing silly walks compared to normal walking. Further studies will have to investigate if the tibialis anterior requires more modulation from supraspinal or other afferent sources during perturbed walking or while performing silly walks.

1. Introduction

Humans and animals are able to perform static, dynamic, explosive and rhythmic movements in an elegant way. Considering that a huge part of our lives involves some form of motion, whether it be walking, cycling, or typing out manuscripts, the neural system uses a considerable amount of computational power to activate muscles to perform our desired tasks (Grillner and Jessell, 2009). Therefore, when neuromuscular diseases or injuries to the musculoskeletal system occur, the motor control system is affected, as it cannot adequately function at its optimum.

The understanding of motor control, especially in upright biped locomotion is challenging. For instance in humans, we have to utilise specific mechanisms to maintain equilibrium on the stance leg during locomotion (Dietz et al., 1986). This is further demonstrated by the difficulties involved in building biped robots (Hirai et al., 1998). A century-old dream was to build machines that can replicate human functions and behaviour. Yet, this has been constantly hindered from complications such as robustness against noise, and flexibility and adaptability of the system, among others (Ijspeert 2006). Yet, there have been many exciting theories in robotics, which can provide a better understanding of human locomotion (Ijspeert 2008). Instead of an active control, McGeer 1990 found that the inherent properties of a mechanical system could produce walking in a passive robot. Taga et al., 1991 demonstrated that synchronizing the neural system with periodic feedback signals resulted in stable locomotion even in an unpredictable environment. Studies by Geyer et al., 2003, and Geyer and Herr 2010 showed that biped locomotion can be solely based on reflexes.

1.1. Hierarchical neural networks

Different control network at different levels of the nervous system contribute to human motor control. Since neural systems are the result of evolution, they follow certain universal rules (Rabinovich et al., 2006); one of which is that neural networks are organised in a hierarchy. Similarly, Duysens et al., 2002 argued that there are different neural control levels, or "spinal robots" in humans. The higher level robots can influence the lower level robots. The lowest basic layer is the one that humans use when walking without any deliberation. A higher layer in the brain would take over during perturbed walking when a higher degree of conscious control is

required. These ordered networks allow us to predict and define motor requirements whether it be performing a specific task, or multi-tasking.

1.2. Spinal patterns generators

The lowest level of neural control which is responsible for generating the basic patterns of locomotion, is believed to come from spinal pattern generators (SPG) located in the spinal cord (Grillner and Wallen, 1985). In this study, we used the term SPG instead of central pattern generators as we specifically refer to neural networks in the spinal cord that do not require a central input from the brain in order to create a motor output. We are specifically referring to the spinal circuitry which is capable of generating locomotion when subjected to tonic or phasic sensory afferents caudal to a complete spinal transection (Rossignol and Frigon, 2011). These neural networks consist of an ensemble of motoneurons whose combined operations will generate the fundamental spatio-temporal patterns of rhythmic movements (Stuart and Hultborn, 2008).

In one of his pioneering work on the origins of spinal stepping, Brown 1911 showed alternating activations between flexor and extensor muscles in the hind limbs of decerebrate cats walking on a treadmill. His demonstration of hind limb activations in other decerebrate rabbits and guinea-pigs also provide indirect evidence that afferent inputs to the spinal cord alone is sufficient in producing coordinated electromyographic (EMG) patterns (Brown 1914). Similar results were also found in other simple vertebrates and invertebrates (Grillner and Wallen, 1985; Marder and Bucher, 2001).

Less is known about the organisation of SPG in higher vertebrates, although studies done on spinal cord injured patients claimed that there are some indirect evidence to its existence. Calancie et al., 1994 presented evidence of a “central -and probably spinal- rhythm generator” responsible for stepping motions in a neurologically *incomplete* spinal cord injured patient. Dimitrijevic et al., 1998 showed that patients with *complete* spinal cord injury could produce locomotor-like activity by applying epidural electrical stimulation to the L2 spinal segment. Furthermore, it was demonstrated that coordinated EMG patterns could be induced in patients with complete or incomplete paraplegia while walking on a treadmill (Dietz et al., 1995). A

recent study by Harkema et al., 2011 showed that a paraplegic subject can perform full-weight bearing and leg movements with tonic epidural stimulation (Harkema et al., 2011). This suggested that SPG responsible for locomotion in humans might be similar to those in cats (Dietz et al. 1992). Overall, these findings support the view that humans may use SPG in generating locomotion. Most importantly, while these studies successfully showed the ability of the SPG in producing a motor output without any interference from the brain, they also demonstrate that the interactions between SPG and sensory inputs are essential in generating a dynamic movement (Baessler 1986). Therefore, a neural network without the appropriate sensory inputs will be artificial, and would not be able to produce the desired motor outcomes. Taga, 1995 had shown that a real-time dynamic interaction between the neural and mechanical system, together with sensory information from the environment can influence the motor output of the lower limbs.

1.3. Brown's half-centre hypothesis

In 1911, Sherrington and Brown reported in the physiological society, observations of scratching in completely de-afferented cats (Brown 1911; Stuart and Hultborn 2008). From these observations, Brown (1911; 1912) hypothesized that a spinal centre was responsible for stepping motion. His idea was radical, since it was a suggestion that stepping movements were generated by spinal reflexes, or has a spinal origin. In actual fact, Brown's collaborator and Noble Prize winner Sherrington was the first to suggest this theory, but had mixed feelings about it. At that time, it was the general consensus that sensory inputs could enhance the quality of rhythmic movements, but the nature of a spinal origin in locomotor pattern generation does not exist. Instead, a cerebral dominance in locomotion had more credence. Still, from Brown's work on spinal guinea-pigs, rabbits and cats, Brown proposed a "half-centre model" which composed of paired but opposing halves; one half will excite the flexors while inhibiting the extensors, while the other half does the opposite (Figure 1). The rhythmic output would be modulated by sensory proprioceptive inputs. Brown's model is important in the understanding of mutually inhibitory connections within the spinal cord, since reciprocal inhibition is of prime importance for generating alternating activation patterns, and its absence may be responsible for pathological gait (Grillner et al., 1998; Knikou and Mummidisetty, 2011).

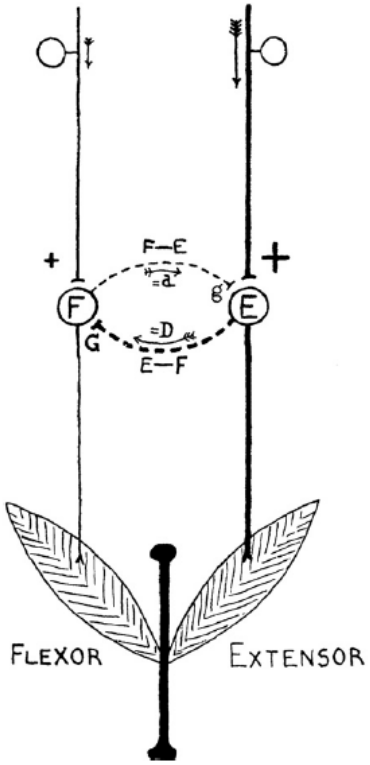

Figure 1: Graham Brown's half-centre model for the control of stepping (Stuart and Hultborn, 2008). Reciprocal inhibition between flexor (F) and extensor (E). Capital D refers to strong inhibition; small d refers to a weaker inhibition. Capital G is fatigue; small g is for less fatigue.

Similarly, mathematical models based on mutual inhibition in neural networks worked on the same theory; when one neuron is activated, the other is suppressed. In these models, each neuron, depending on the input received, can alter the rhythmic pattern and therefore, may be applied to describing rhythmic motion in humans and animals (Matsuoka 1987). The concept of neural oscillators or networks has been applied to the control of two or multi-legged walking models (Taga et al. 1991, Ijspeert and Kodjabachian 1999, Maufroy et al. 2008; Sussilo and Abbott 2009). While Maufroy et al. 2008 utilized limb loading as a sensory-triggered control during stepping for quadruped locomotion, some models were centrally controlled (Ijspeert 2008).

1.4. Supraspinal influences

Animal locomotion was studied in detail for a very long time. It was the accepted belief that a cerebral dominance, rather than spinal mechanisms is responsible for locomotion. For instance,

higher-centres are necessary for modulating locomotor patterns in cats (Armstrong 1988). In many vertebrae animals, stimulation of certain areas of the brain stem for example, at the Mesencephalic locomotor region has been known to induce more complex movements including fictive locomotion (Amemiya and Yamaguchi, 1984). However, less is known about the central control during walking in man. Obviously, this is due to ethical issues involving invasive, experimental procedures to the brain of a living, breathing human subject. So, the role of the brain in normal human walking is gathered chiefly from patients suffering from brain lesions, brain imaging studies and electrophysiological studies (Nielsen 2003). In addition, most of the understanding of supraspinal control in humans mainly rests on the assumption that the principles governing motor control during human walking are similar to other animals. Yet, human walking is unique; we walk on two legs, while the chimpanzees, our closest relative choose to walk on fours. It was Aristotle who suggested that since man has the largest brain among animals, it allows us to stand erect. Though he made the (albeit incorrect) conclusion that the heart, rather than the brain, controlled sensation and movement, he was one of the first philosophers to suggest the importance of the brain in enabling man to acquire a more superior neural control in bipedalism.

Still, there has been no evidence that the brain is solely responsible for activating the muscles during walking, or that the contribution from neural networks in the spinal cord is negligible (Nielsen 2003). It is highly likely that integration between supraspinal control and the spinal circuitries, with sensory feedback signals, is crucial in the unique walking patterns seen in humans.

1.5. Sensory afferents

Afferents are continuously interacting with different parts of the nervous system. As the human nervous system should efficiently coordinate, respond and adapt to the immediate environment, it is important that the plethora of signals coming from the central, sensory and peripheral systems be selected and modulated, such that the motor output fulfils the demands of the locomotor task.

While spinal networks potentially use a wide range of sensory afferents to generate muscle activity (Dietz and Duysens 2000), it has been difficult to implicate specific signal inputs responsible for the control or modification of muscle activity at certain phases of gait. It has also been difficult to determine when cortical inputs would dominate over underlying reflex actions in an adult man with an intact nervous system (Duysens 2002). However, studies on infants (Pang and Yang 2000) and patients with complete spinal cord injuries (Dietz et al. 2002) identified two main afferents. Load receptors, consisting of both mechanoreceptors at the soles of the feet and proprioceptive receptors in the muscles, and hip joint-related afferents seemed to be essential in generating locomotor-like activity in humans. During the stance phase, load receptors increase extensor activity while suppressing the onset of the swing phase. At the end of stance, hip extension is a signal for the start of the swing phase.

1.6. Loading afferents

The importance of loading receptors for locomotion had been studied in cats (de Guzman et al., 1991; Gorassini et al., 1994, Duysens et al., 2000) and humans (Dietz et al., 1992; Harkema et al., 1997; Dietz and Duysens, 2000). Dietz et al., 1995 particularly pointed out the importance of unloading and reloading paraplegic patients during training sessions. It is an important part of rehabilitation as this may serve as a stimulus for load receptors in muscles, and allows the patients to achieve locomotor-like muscle activation.

Yet, different studies have reported different outcomes to the loading and unloading response performed on cats and humans. For the tibialis anterior, Harkema et al., 1997 found a significant relationship between the mean EMG amplitude of the tibialis anterior and loading during stepping. However, af Klint et al., 2009 found that loading has little or no effect on the activity of the tibialis anterior. For the soleus, previous studies suggested a close relation between loading and ankle extensor activity (Gorassini et al., 1994; Sinkjaer et al., 2000; Donelan et al., 2004; af Klint et al., 2009). The authors found that an increase in loading resulted in an increased activity in the soleus, and vice versa. However, Nakazawa et al., 2004 found an increase in soleus activity when there was a loss of ground support. These differences could be due to the fact that a loss of one afferent could trigger different compensatory reactions.

1.7. Aims

First, we developed a simple two-neuron SPG model, and validated the model with experimental data from normal walking at self-selected speed. Loading and hip orientation were used as inputs into the model, which would be applied to the ankle joint. Our first aim is to demonstrate that rhythmic motion at the ankle can be explained by an SPG model, which is only triggered by sensory afferents without any interference from a cortical signal. This might also show that SPG provide a direct link between sensory inputs and muscle activations via motoneurons.

After the first aim was accomplished, we wanted to determine whether neural networks in the spinal cord can adapt to changing sensory cues, and directly influence muscular activity to meet the motor demands of different types of walking patterns. So, we studied the response of the SPG model, which is only triggered by sensory afferents, in three different situations where spatio-temporal parameters in a gait cycle will change: -

<u>Case 1 – Sudden loss in loading afferents</u>

Since the organisation of neural networks is hierarchical, a compromised motor control system due to a loss in sensory afferents can also be taken over by another layer of neural control. This replacement could be performed since bipedal locomotion extends to the entire spinal cord from cervical to lumbar levels (Dietz et al., 1999). In addition, the brain could intervene. The question is whether the motor changes from a loss of afferent inputs can be performed at the spinal level, or require intervention from supraspinal inputs.

<u>Case 2 – Change in walking speed</u>

While we know that sensory afferents can influence muscular activations, is the same neural network involved when motor demands change? Walking at a slower or faster pace creates different motor demands on the neural system. A number of gait components such as stance and swing phase intervals, and muscle activation patterns change with increasing speed. However, in healthy humans, it is not known whether these changes are a result of a common set of neural oscillators in the spinal cord, a separate additional set of oscillators for different gait patterns, or higher commands from supraspinal areas.

Case 3 – Silly walks

Silly walks are just that – silly. Check out Monty Python's sketch, The Ministry of Silly Walks. While this sketch is hilarious, the movements are erratic and awkward. Since a healthy neuro-musculoskeletal system runs elegantly and optimally, erratic walking motions would be an inefficient way to move from point A to point B, unless conscious effort was made to perform a silly movement. So, we hypothesize that the SPG model will show significant differences in the model parameters because the silly walks are performed due to a command from the brain.

2. Methods

The methods consist of three parts; first, we fit the outputs from the SPG model to experimental EMG data. This was performed for every trial for all walking situations. Second, we introduce a perturbation by a sudden removal of the loading input while retaining hip angles in the SPG model, and calculated simulated EMG responses. This was only performed for normal walking trials at self-selected speed. Third, we collected experimental EMG data during sudden unloading in four subjects, and compared these to the simulated EMG responses.

2.1. Experimental measurements

Seven male healthy subjects (28.0±4.4 years, 1.8±0.1 m, 76.4±9.5 kg) volunteered to participate in this study. They were thoroughly informed of the procedures and gave their consent. Each subject was requested to walk at his normal self-selected speed (4.8±0.5 km/h), at 3.5 km/h, 4.0 km/h and 4.5 km/h on a treadmill (Kinetics s3, Kettler, Germany). In addition, they were asked to perform movements unlike their normal walking i.e. "silly walks" at a speed of their own choosing (3.8±0.4 km/h). Data from six consecutive strides was collected during steady-state walking. Three trials were recorded for each subject for each walking speed and silly walks i.e. a total of 210 trials (for both right and left limbs). Trials were removed if there were missing kinematic or EMG data in any one stride. Therefore, only a total of 176 trials was analysed in this study.

Force data was collected at 200Hz, and calculated from in-shoe pressure sensors (Gesellschaft für Biomechanik Münster, Germany) as a summation of pressure acting on the entire area of the insole. Hip angles were acquired from Oqus 3D motion analysis system (Qualisys, Gothenburg, Sweden) at 100 Hz. The motion analysis system used six infra-red cameras which tracked a total of fifteen retro-reflective markers attached to the following body landmarks: lateral and medial knee and four tracking markers on the thigh of each leg, sacrum, left and right anterior superior iliac spine. Segment definitions and kinematic data were processed using Visual3D (C-Motion Inc, Maryland, USA). Muscle activation from the soleus (SOL) and tibialis anterior (TA) (Figure 2) were captured using bipolar surface electrodes (5–700 Hz, Biovision, Wehrheim, Germany) at 2000 Hz. The SOL and TA muscles were chosen because they are the principal monoarticular plantarflexor and dorsiflexor muscles, respectively. Electrodes were placed according to

recommendations by Hermens et al. 1999. The EMG signals were centred, rectified and filtered using a fifth-order low-pass Butterworth filter with a cut-off frequency of 40Hz. Pressure and EMG systems were synchronized using an internal trigger; Pressure measurements started when the pressure system detected an EMG input. Kinematic and EMG system were synchronized using an external trigger; Kinematic and EMG measurements began when a 'start' pulse was detected. So, all measurements were captured simultaneously with a 'start' signal.

2.2. Model

A simple Matsuoka oscillator (Matsuoka, 1985; 1987) consisting of two neurons was used; one neuron will activate the SOL and the other will activate the TA (Figure 3). Thus, the outputs from the oscillator represented the corresponding muscle activation of each muscle. The neurons are mutually inhibited, i.e. when one neuron is activated, the other is suppressed.

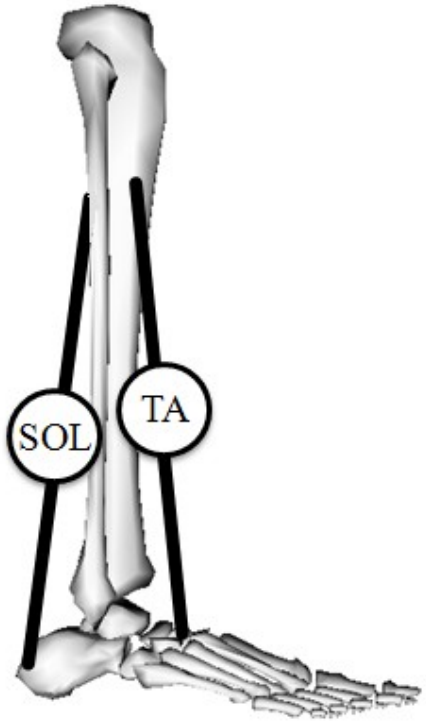

Figure 2: An ankle joint with soleus (SOL) and tibialis anterior (TA) muscles.

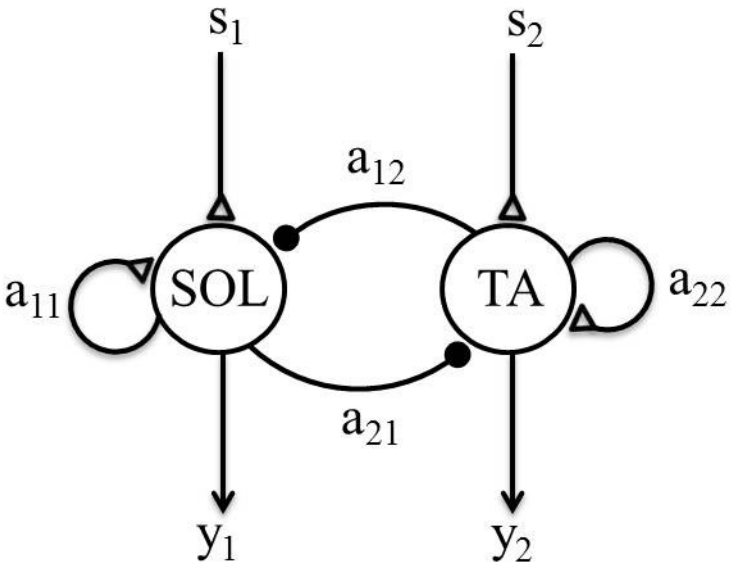

Figure 3: SPG model consisting of two neurons (SOL – Soleus, TA – Tibialis Anterior). Triangles represent excitatory signals/connections, dark spheres represent inhibitory connections. Subscript 1 refers to SOL, and subscript 2 refers to TA.

The oscillator is governed by the following equations (adapted from Matsuoka 1985):

$$\dot{x}_i + x_i = \sum_j a_{ij} y_j + s_i - b_i f \qquad (1)$$

$$T_i \dot{f}_i + f_i = y_i \qquad (2)$$

$$y_i = \max(0, x_i) \qquad (3)$$

where f is the adaptation in the neuron, T and b are the parameters that determine the time course of the adaptation. When b=0, there is no adaptation, and the output will increase and then remain at a constant value (Refer to Figure 1 in Matsuoka 1985). x is the inner state of the neuron, y is the generated output of the neuron, s is the input signal, and a is the strength of the connection between both neurons; a_{ij} <0 for $i \neq j$ (mutual inhibition) and >0 for $i = j$ (self-excitation). We assume a symmetrical arrangement of the neurons, i.e. $a_{ij} = a_{ji}$, $a_{ii} = a_{jj}$.

Vertical force calculated from the insoles was first normalised to the subject's weight. Normalised force *F* and hip flexion/extension angles *HA* of the ipsilateral limb (in radians) were used to determine the signal input s_i in equation (1):

$$s_i = m_i.p + n_i.\dot{p} + w_i.q + v_i.\dot{q} \quad (4)$$

with

$$\dot{p} = r_1(F - p) \quad (5)$$

$$\dot{q} = r_2(HA - q) \quad (6)$$

where *r* is the smoothing coefficient, *p* and *q* are the smoothed *F* and *HA* respectively, and $\dot{p}$ and $\dot{q}$ are the respective change in *F* and *HA*. *m*, *n, w* and *v* represented weights of each excitation *p*, $\dot{p}$, *q*, $\dot{q}$ respectively. Therefore, this model is triggered by both the magnitude and the change in magnitude of loading and hip angles.

2.3. Fitting

The parameters *a, b, m, n, r, T, v, w* from the above equations, determined the pattern and frequency of the output. Since the parameters had to be selected and tuned (Williamson 1999; Ogihara and Yamazaki 2001), a trust-region nonlinear least-squares (NLS) fitting algorithm was used to determine a set of parameters that would fit the output to experimental data for each trial i.e. the output produced by the neuron representing the TA would be fitted to measured EMG of the TA. This was done similarly for the SOL. During fitting of each trial, initial values for each neuron were taken from the first value of measured EMG data from that respective trial, so as to numerally solve the differential equations. The fitting algorithm was terminated once the relative deviation between two iterations fell below 0.001. A correlation coefficient *R* between the model output and experimental EMG data was calculated for each trial after fitting.

In addition, a 95% confidence bound was also determined from the root mean squared error of each parameter. This confidence bound gave the lower and upper values of each parameter before the output deviated 5% away from best fit. The range of these values may indicate how sensitive the fitting is to the parameters. A smaller range meant that the output is more sensitive to the parameter since a slight deviation from best fit value would result in a poorer fitting. A

wider range may indicate that the parameter could not be precisely determined, either due to the model, or that more data is required for fitting of that particular parameter.

The following gait components were analysed; Maximum normalised force and maximum range of hip flexion-extension angles were calculated for each stride. Stance and swing phases determined from force profiles of each stride were also calculated. For these gait components, analysis of variance (ANOVA) and Tukey's post-hoc test were performed to determine the significant differences between all the different walking types. In analysing the rectified EMG signals for each subject, cumulative numerical integration (IEMG) for each EMG signal in each stride was calculated for all speeds. The maximum of the mean IEMG values was designated as 1.00, regardless of speed. The other mean values were normalised with respect to this maximum value. To determine significant differences in the model parameters ($p<0.05$), multivariate analysis of variance (MANOVA) along with analysis of variance (ANOVA) and Tukey's post-hoc test were performed.

2.4. Perturbation in model

A perturbation was introduced into the SPG model by a sudden removal of the loading input at certain instants during stance during the sixth (last) stride. This represents a situation where a sudden loss of ground support occurs during walking, for example when a person experiences a sudden loss in footing while stepping into a hole. Four instants during the last stride were identified for each trial using insole forces: heel strike (HS), first peak or loading response (1P), midstance (MS) and second peak or terminal stance (2P) (Figure 4). A removal, rather than the addition of loading inputs, was performed because previous experimental studies have argued that an "unload response" is more appropriate for the study of loading afferents, rather than enhancing the afferent input to the spinal network (Nielsen and Sinkjaer, 2002).

IEMG was calculated during the 100ms period after the introduction of the perturbation. We did so because it was found that duration of a response from a perturbation was within the 100ms window (Grey et al., 2007; af Klint et al., 2009). One-way analysis of variance (ANOVA) and Tukey's post-hoc test were performed to determine the significant differences ($p<0.05$) between the normal and perturbed conditions.

2.5. Perturbed experiments

The perturbation experiments were performed in Center For Sensory Motor Interaction, Aalborg University, Denmark. Four male subjects (36.8±8.5 years, 1.7±0.1 m, 76.0±18.1 kg) volunteered in these perturbation trials. All subjects walked barefoot along a 10-m pathway across a robotic platform with a force platform mounted on top (OR6-5, Advanced Mechanical Technology, Watertown MA) that is flushed with the floor (van Doornik and Sinkjaer, 2007). This robotic platform utilised hydraulically actuated pistons to drop vertically by 4 cm with a constant acceleration and deceleration of +/-0.8g. The latency timings, at which the movement of the platform was initiated, were determined prior to the perturbation trials by requesting the subjects to walk normally over the platform. The force profiles of five normal walking trials were averaged, and the latency timings corresponding to the four instants at HS, 1P, MS and 2P (as presented in Figure 4) were determined.

One set of measurements consisting of 24 trials were randomly presented to each subject. Three trials were performed for each perturbation type (Figure 4) such that a total of 12 trials included a perturbation. The other 12 were non-perturbed trials. All trials were randomly ordered to prevent subject anticipation. A total of two sets of perturbation trials were performed by each subject.

SOL and TA muscles of the preferred leg were measured using bipolar surface EMG (5–700 Hz, Biovision, Wehrheim, Germany) at 2048 Hz. The EMG signals were centred, rectified and filtered using a fifth-order low-pass Butterworth filter with a cut-off frequency of 40Hz.

IEMG was calculated during the 100ms period after the introduction of the perturbation. One-way repeated-measures ANOVA test was performed to determine the significant differences ($p<0.05$) between normal and perturbed conditions.

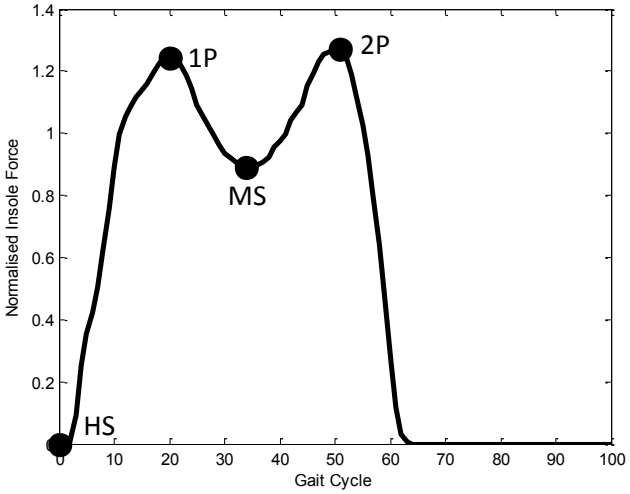

Figure 4: Four instants (dark spheres) were identified for each trial using normalised insole forces. In the SPG model, they represent the instants at which loading was suddenly removed. For the perturbation experiments, they represent the instants at which the platform movement was initiated. HS: heel strike, 1P: first peak (loading response), MS: trough (midstance), 2P: second peak (terminal stance).

3. Results

3.1. Normal walking at self-selected speed

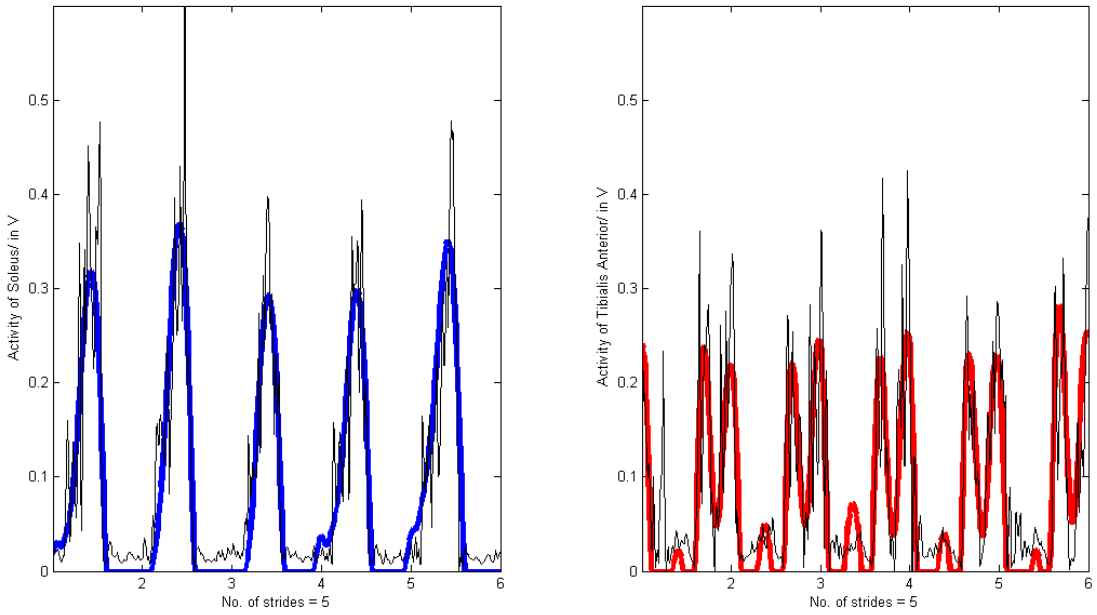

Figure 5: $R_{average(ave)}$=0.85. Muscle activation of the soleus (R_{SOL}=0.90) and the tibialis anterior (R_{TA}=0.80) of one subject walking at a self-selected speed of 3.9km/h with insole forces and hip angles as inputs (bold coloured lines represent the output from the SPG model, thin black lines represent the experimental EMG data)

It was found that the output became oscillatory only after the first stride, and therefore, the results presented in Figures 5-6, 11-13 are only from stride two onwards. Using loading and hip angles as inputs, the SPG model was able to generate outputs close to experimental EMG data. The fitted parameters for all trials are shown in Table 1. The mean correlation coefficient *R* for all walking trials at self-selected speed is 0.79±0.03. The trial with the best fitting of *R*=0.85 is shown in Figure 5. The worst correlation calculated for the TA was R_{TA}=0.59 (Figure 6). The fitting of the other 29 trials for normal walking at self-selected speed would fall between Figures 5 and 6. The SOL had a stronger correlation (R_{SOL}=0.85±0.04) than the TA (R_{TA}=0.73±0.06). The lower correlation appeared to come from TA activation during swing. A possible reason was that the SOL is only active during stance, and peaks approximately between 40-60% of gait, so it is active once every stride, and has a uniform oscillation. On the other hand, the TA is active at

early stance and during swing, so the lower correlation for the TA could be due to a less uniform oscillation. An average of the range between the lower and upper bounds determined at 95% confidence interval is also shown in Table 1. *T* had the smallest range, while *v* had the widest.

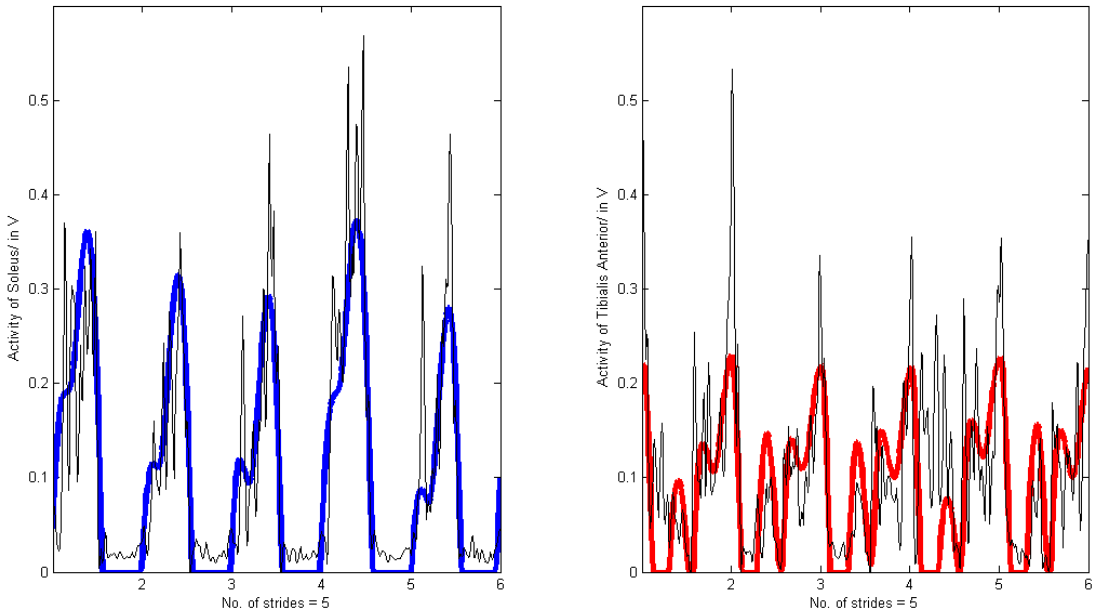

Figure 6: R_{ave}=0.73. Muscle activation of the soleus (R_{SOL}=0.87) and the tibialis anterior (R_{TA}=0.59) of one subject walking at a self-selected speed of 5.1km/h with insole forces and hip angles as inputs (bold coloured lines represent the output from the SPG model, thin black lines represent the experimental EMG data)

parameters	mean ± std	confidence bound
m_1	0.13 ± 1.38	0.24
m_2	0.21 ± 1.82	0.30
n_1	-0.79 ± 0.68	0.15
n_2	-2.24 ± 2.09	0.41
w_1	-3.07 ± 2.18	0.40
w_2	-3.99 ± 3.30	0.59
v_1	-0.45 ± 4.90	5.01
v_2	-9.32 ± 18.08	12.17
r_1	2.74 ± 1.53	0.41
r_2	6.63 ± 9.04	1.24
a_{11}, a_{22}	10.23 ± 7.00	2.73
a_{12}, a_{21}	-3.26 ± 2.84	0.45
T_1	0.12 ± 0.08	0.04
T_2	0.03 ± 0.04	0.01
b_1	15.62 ± 6.93	2.32
b_2	20.64 ± 10.88	3.03

Table 1: Mean, standard deviation and range of confidence bound of fitted parameters for all normal walking trials at self-selected speed. Refer to Figure 3; subscript 1 refers to SOL, subscript 2 refers to TA.

3.2. Case 1 – Sudden loss in loading afferents

When a sudden loss of loading input was introduced into the SPG model, an immediate increase in SOL and TA occurred (Figure 4, Tables 2-3). An example from one representative trial is shown in Figure 7, where a sudden loss in loading was applied during terminal stance. Only the areas of response within the 100ms window after the perturbation were analysed (Tables 2-3). TA showed significant increase in activation patterns in response to loss of loading during 1P, MS and 2P. SOL only showed significant difference between normal and perturbed conditions during 1P.

In the perturbation trials, significant difference in the SOL was only found during the loading response (Table 2), which was in line with our model. For the TA, significant differences were only found during midstance (Table 3). A set of data from one subject is shown in Figures 8-9.

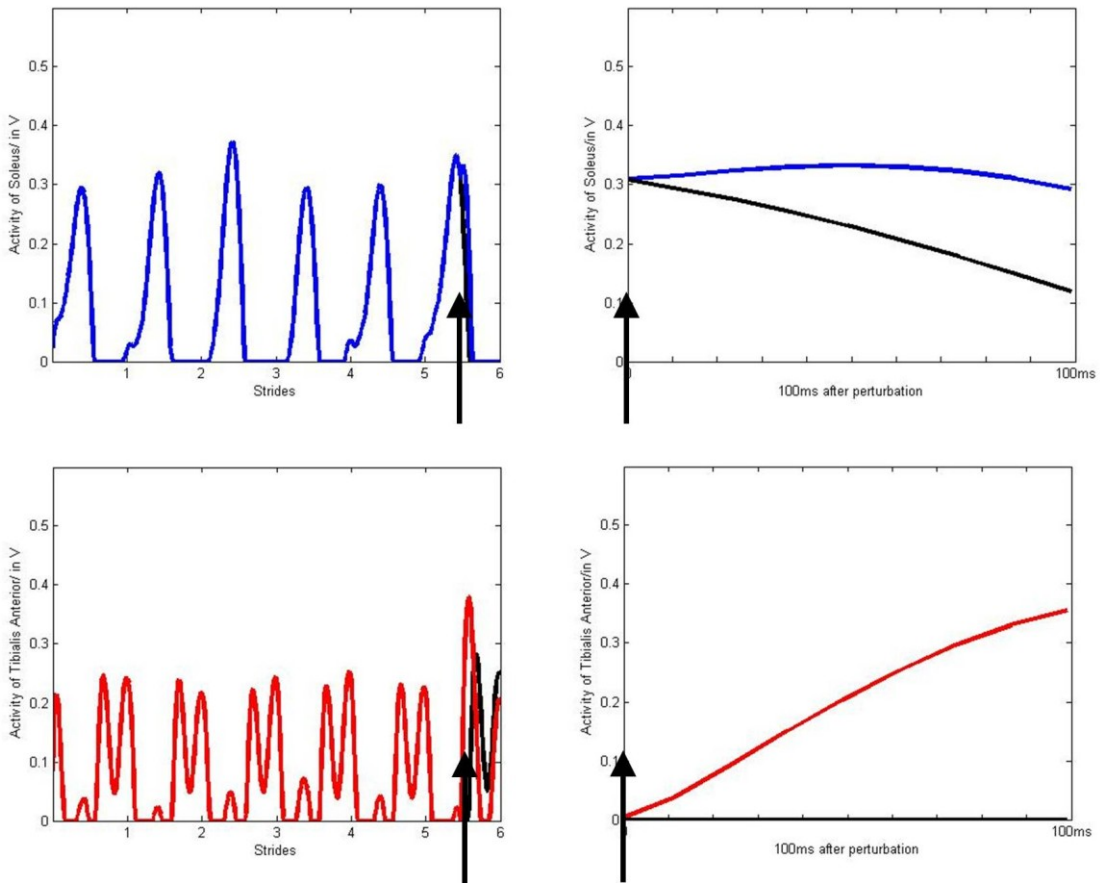

Figure 7: Generated muscle patterns of the soleus (top row) and tibialis anterior (second row) when perturbation was introduced at terminal stance (2P in Figure 4) or terminal stance during the sixth stride (left column). On closer look (right column): during the 100ms window after perturbation occurred. Black lines represent EMG during normal walking and coloured lines represent EMG after sudden loss in loading input at terminal stance. Arrows signify start of perturbation.

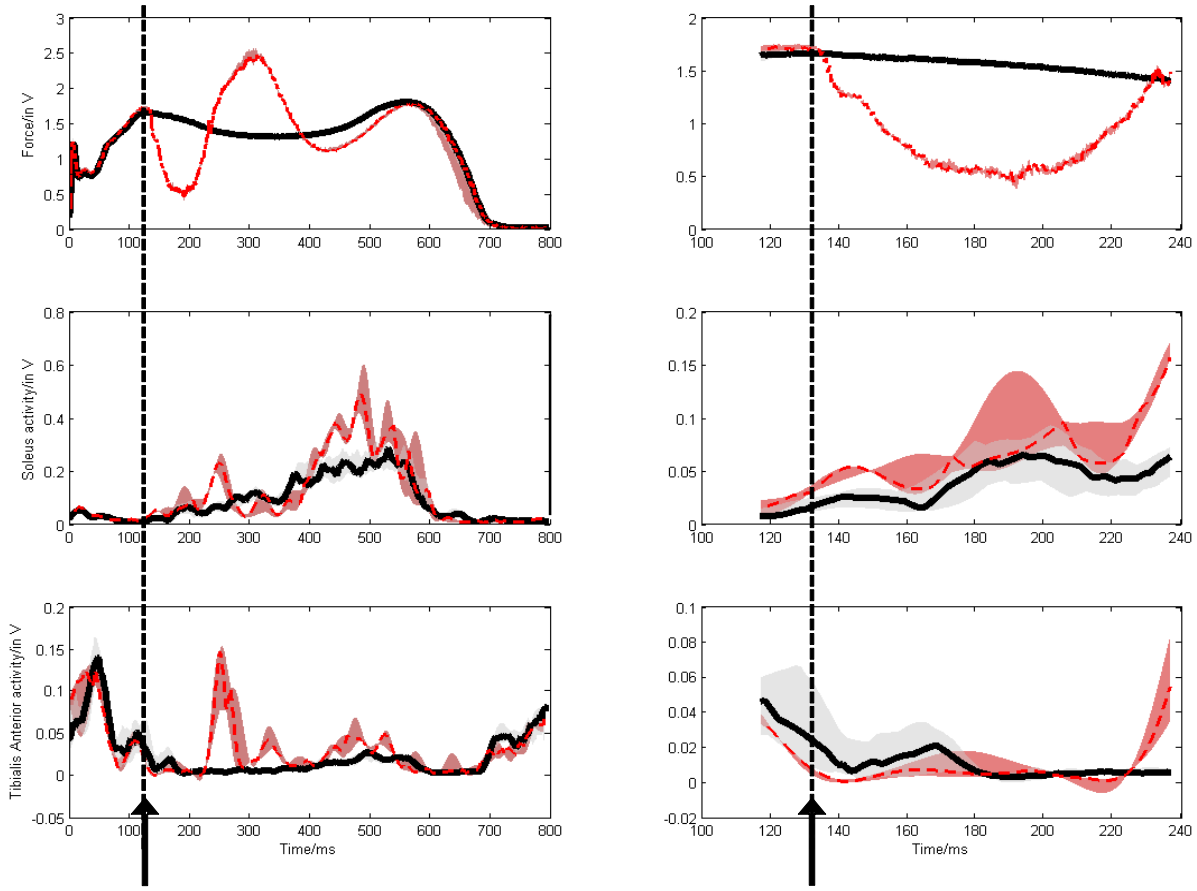

Figure 8: Averaged perturbed trials from one set of measurements of one subject when sudden loss of ground support was introduced at first peak (1P in Figure 4). On closer look (right column): during the 100ms window after perturbation occurred. Thick black lines represent force or EMG during normal walking (n=12) and dashed red lines represent force and EMG after sudden loss of ground support at 1P (n=3). Arrows signify start of perturbation. Here, an increase activity in SOL was demonstrated. However, there was no significant differences in the TA.

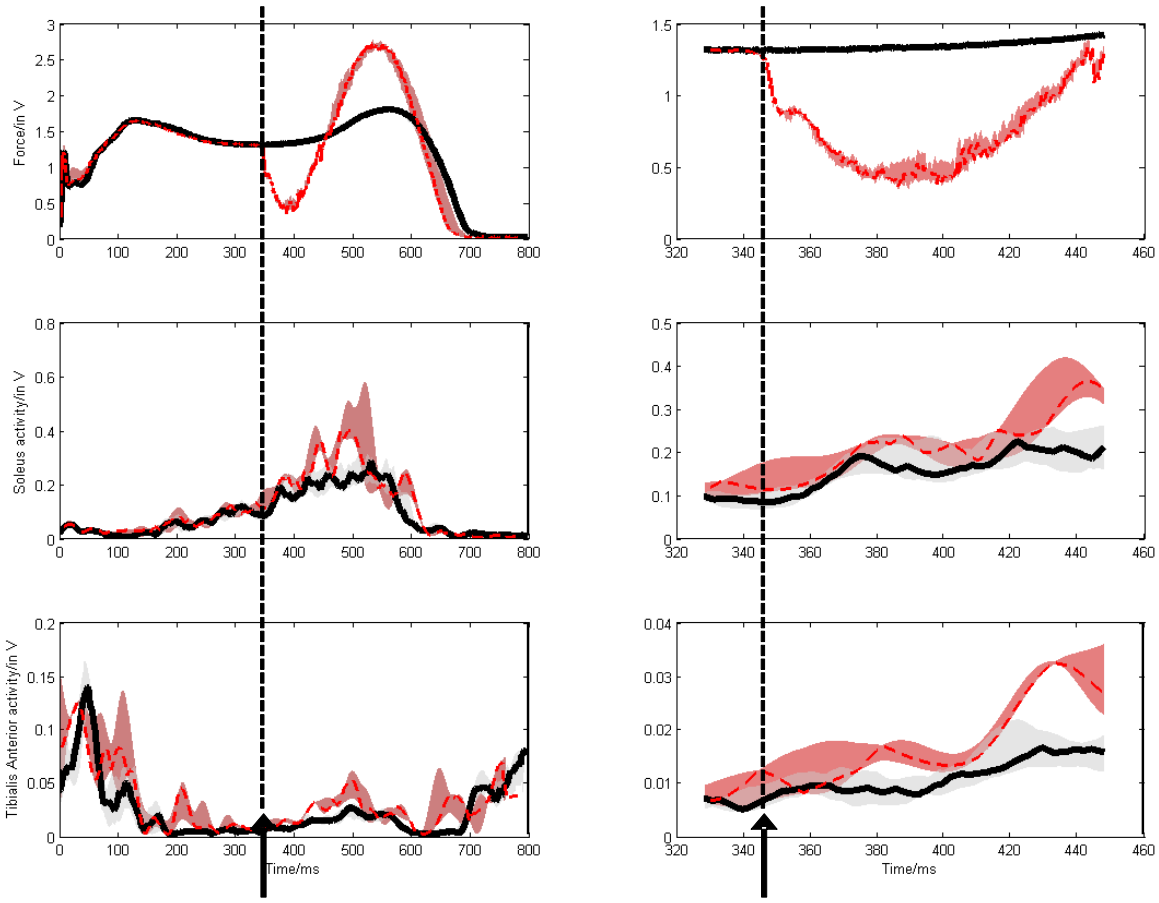

Figure 9: Averaged perturbed trials from one set of measurements of one subject when sudden loss of ground support was introduced at midstance (MS in Figure 4). On closer look (right column): during the 100ms window after perturbation occurred. Thick black lines represent force or EMG during normal walking (n=12) and dashed red lines represent force and EMG after sudden loss of ground support at MS (n=3). Arrows signify start of perturbation. Here, an increase activity in the TA was demonstrated. There was no significant differences in the SOL.

SOL-model						SOL-perturbed trials				
	Normal		Perturbed		p	Normal		Perturbed		p
	mean	±std	mean	±std		mean	±std	mean	±std	
HS	5.75	±3.90	8.23	±5.30	NS	3.39	±2.20	3.75	±2.09	NS
1P	10.69	±4.38	23.84	±8.30	<0.05	7.06	±4.40	9.27	±5.44	<0.05
MS	30.67	±8.08	38.76	±12.77	NS	14.01	±9.42	16.44	±9.55	NS
2P	14.48	±7.33	22.41	±10.68	NS	16.16	±14.67	15.58	±12.95	NS

Table 2: Mean and standard deviation (std) of IEMG (in mV s) of the SOL calculated in the SPG model (left) and in the perturbation trials (right). SOL activity during the 100ms period after perturbation was introduced at heel strike (HS), first peak (1P), midstance (MS), second peak (2P) for normal and perturbed walking. p describes the significant difference between normal and perturbed conditions. NS: not significant.

TA-model						TA-perturbed trials				
	Normal		Perturbed		p	Normal		Perturbed		p
	mean	±std	mean	±std		mean	±std	mean	±std	
HS	16.15	±6.48	25.05	±13.72	NS	11.46	±6.25	10.04	±4.89	NS
1P	0.75	±0.97	8.85	±4.68	<0.05	1.82	±1.85	2.04	±1.78	NS
MS	3.96	±4.20	22.83	±13.66	<0.05	1.68	±0.93	2.46	±1.15	<0.05
2P	2.26	±1.93	26.57	±19.91	<0.05	4.06	±3.02	4.00	±2.05	NS

Table 3: Mean and standard deviation (std) of IEMG (in mV s) of the TA calculated in the SPG model (left) and in the perturbation trials (right). TA activity during the 100ms period after perturbation was introduced at heel strike (HS), first peak (1P), midstance (MS), second peak (2P) for normal and perturbed walking. p describes the significant difference between normal and perturbed conditions. NS: not significant.

3.3. Cases 2 & 3 – Change in walking speed and silly walks

There were no significant differences found for *R* between normal walking at self-selected speeds and walking at other speeds. However, *R* calculated for silly walks (mean correlation R_{mean}=0.70±0.08) were significantly different to the other walking types (Figure 10). So, the quality of the fitting for silly walks is not as good as the other walking types (examples of three trials of one subject are presented in Figures 11-13). It is also possible that the sensory inputs used in the model were insufficient to account for the muscular activations measured. Here, it is unknown if additional sensory inputs or a cortical signal would give a better correlation.

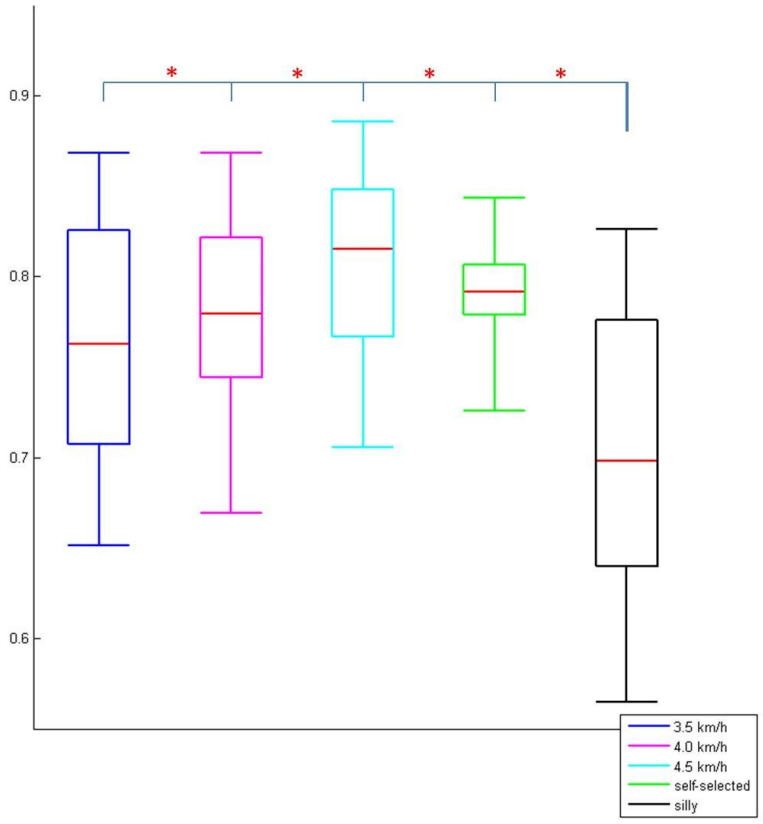

Figure 10: Correlation coefficient *R* values at different speeds and silly walks. The tops and bottoms of each box are the 25th and 75th percentiles of *R* respectively. Red lines are the median values. (*) denotes significant difference.

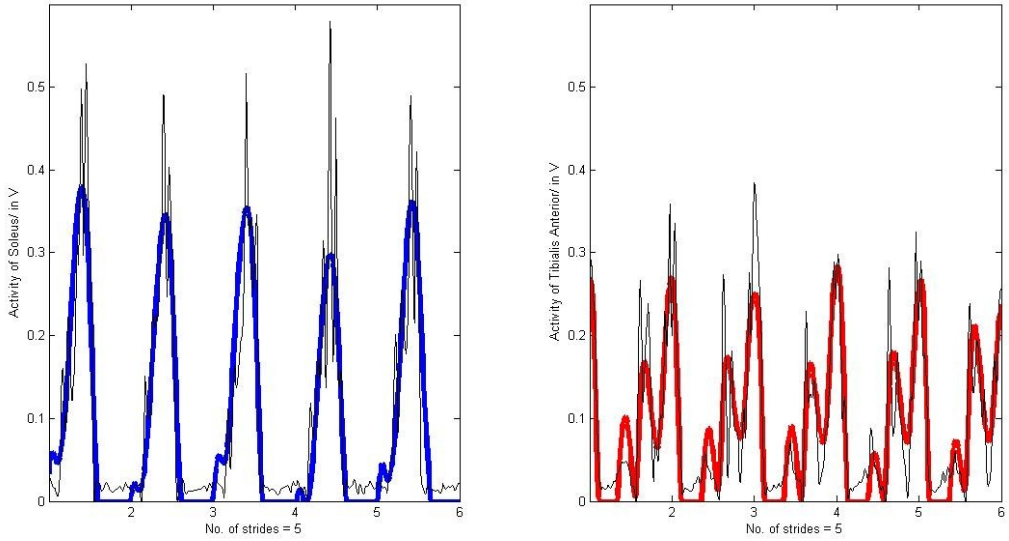

Figure 11: R_{ave}=0.88. Muscle activation of the soleus (R_{SOL}=0.90) and the tibialis anterior (R_{TA}=0.87) of one subject walking at 4.5km/h with insole forces and hip angles as inputs (bold coloured lines represent the output from the SPG model, thin black lines represent the experimental EMG data).

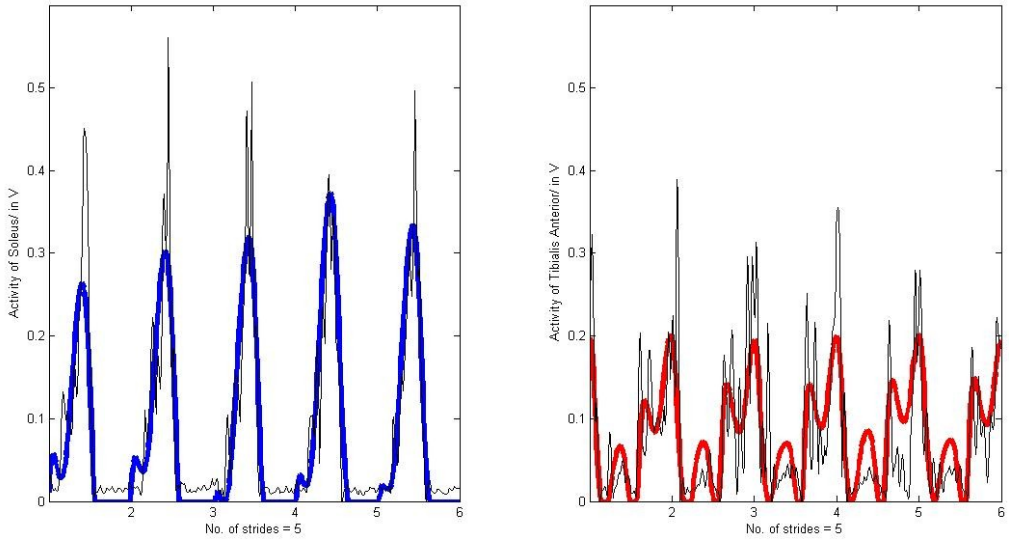

Figure 12: R_{ave}=0.80. Muscle activation of the soleus (R_{SOL}=0.90) and the tibialis anterior (R_{TA}=0.71) of one subject walking at 4.0km/h with insole forces and hip angles as inputs (bold coloured lines represent the output from the SPG model, thin black lines represent the experimental EMG data).

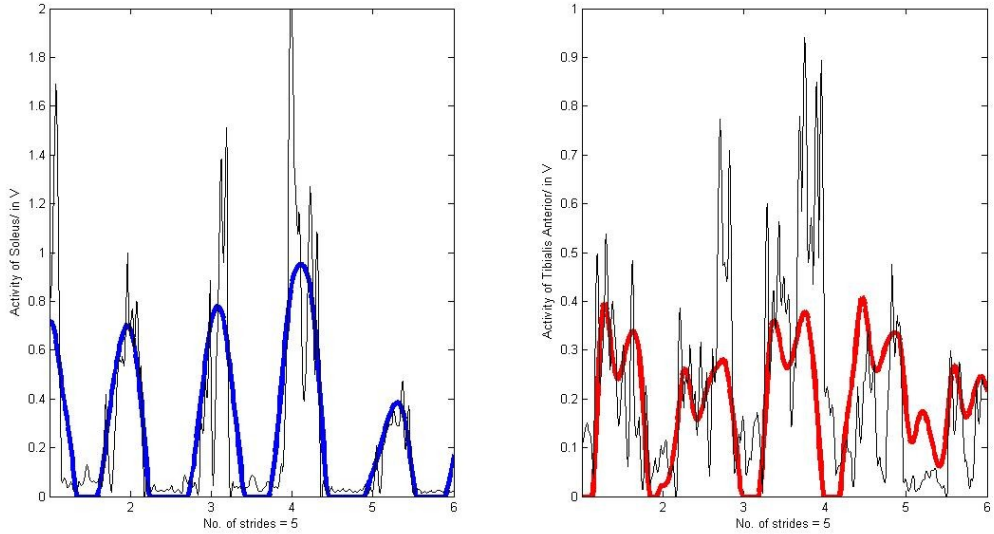

Figure 13: R_{ave}=0.63. Muscle activation of the soleus (R_{SOL}=0.79) and the tibialis anterior (R_{TA}=0.46) of one subject performing silly walks with insole forces and hip angles as inputs (bold coloured lines represent the output from the SPG model, thin black lines represent the experimental EMG data).

Significant differences were found in the gait components calculated (Table 4). As expected, an increase in walking speed is demonstrated by a decrease in the relative stance phase duration, an increase in the relative swing phase duration, an increase in hip flexion-extension angles, and increase in the peak values of the SOL and TA (Murray et al., 1984). Since loading and hip angles were significantly different, this meant that inputs to the SPG model were significant differently for all walking types.

MANOVA reported significant differences between the model parameters. To continue with the analysis, ANOVA followed by Tukey's post-hoc test reported no significant differences found in all model parameters for normal walking at self-selected speeds and other speeds (Figure 14). Only T_2, the time constant responsible for the time lag of the adaptation effect in the TA showed significant differences between the silly walks, and the other normal walking trials.

	3.5 km/h		4.0 km/h		4.5 km/h		self-selected 4.8±0.5 km/h		silly walks 3.8±0.4 km/h		p
	mean	std	mean	std	mean	std	mean	std	mean	std	
Stance (%)	66.60	±4.85	65.59	±4.34	65.22	±4.48	64.99	±1.58	63.33	±7.34	$p<0.05$
Swing (%)	32.97	±3.01	34.05	±2.70	34.38	±2.79	35.01	±1.58	36.67	±7.34	$p<0.05$
Hip flexion-extension range (rad)	0.67	±0.21	0.73	±0.22	0.76	±0.26	0.77	±0.07	0.84	±0.32	$p<0.05$
Max F	1.11	±0.19	1.17	±0.22	1.23	±0.25	1.26	±0.20	1.25	±0.29	$p<0.05$
SOL	0.47	±0.26	0.51	±0.27	0.53	±0.31	0.53	±0.20	0.69	±0.20	$p<0.05$
TA	0.42	±0.26	0.47	±0.28	0.51	±0.32	0.53	±0.21	0.65	±0.21	$p<0.05$

Table 4: Mean and standard deviation of gait components at different speeds

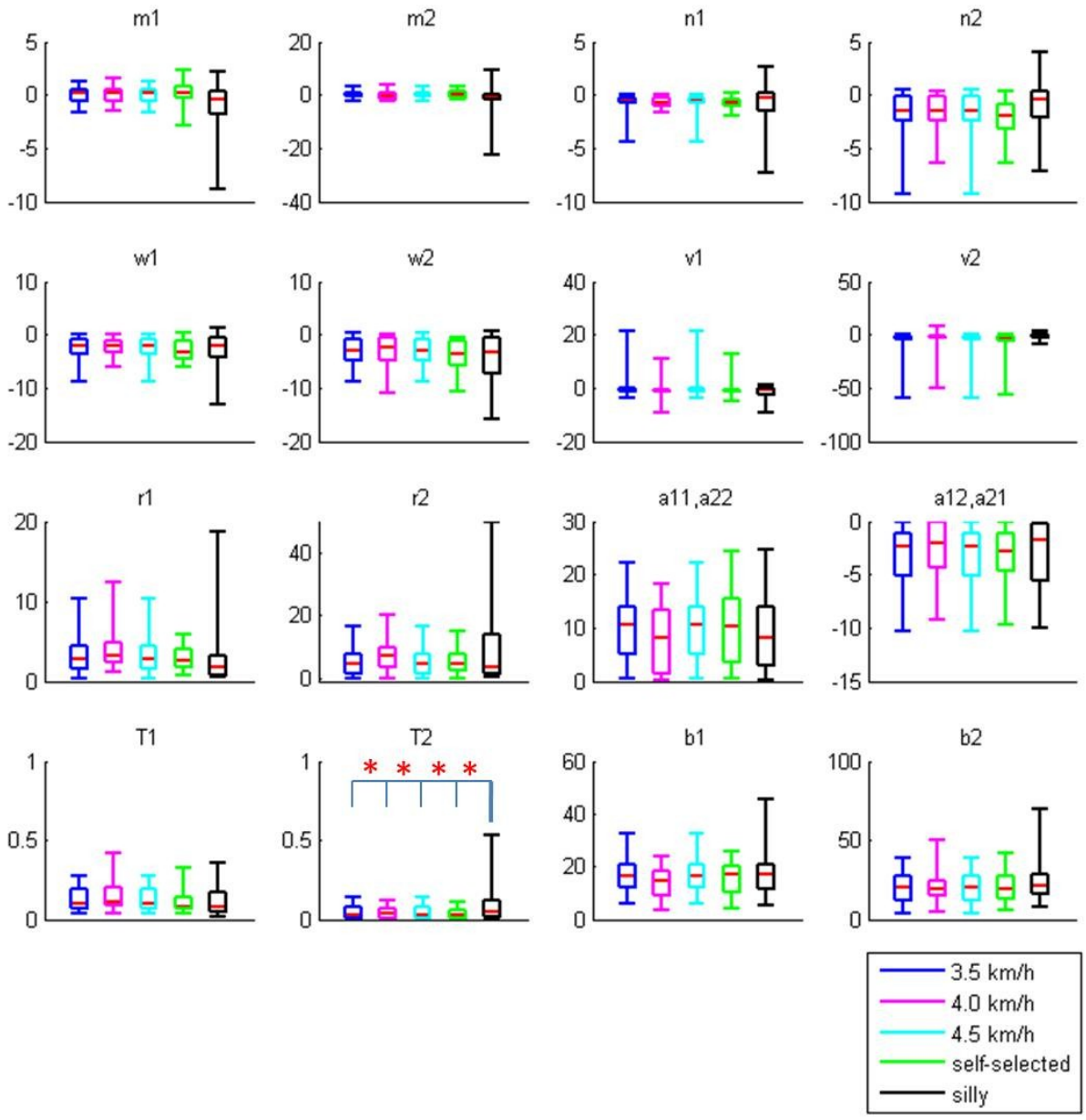

Figure 14: Values of all parameters at different speeds and silly walks. The tops and bottoms of each box are the 25th and 75th percentiles of the parameters respectively. The whiskers represent the range of values. Red lines are the median values. Refer to Figure 3; note that subscript 1 refers to SOL, subscript 2 refers to TA. (*) signifies significant difference.

4. Discussion

These results showed that neural network responsible for muscle activation at the ankle can process different sensory cues to generate stepping motion during normal steady-state walking. In healthy subjects, it is difficult to determine whether the elevated EMG patterns are from supraspinal control, a result of activations from sensory inputs, or an interaction from both supraspinal and spinal control. However, in our model which only consists of spinal neurons, the outputs generated by our model suggest that muscle activations can be generated by sensory inputs from loading and hip angles at the spinal level.

4.1. Normal walking at self-selected speed

4.1.1. Model

The Matsuoka oscillator (Matsuoka 1985; 1987), which consists of mutually inhibiting neurons described by a set of differential equations (equations 1-3), has been widely used in locomotion-related models (Taga et al., 1991; Kimura et al., 1999; Ogihara and Yamazaki 2001; Ishiguro et al., 2003). While there are other oscillators, the popularity of the Matsuoka oscillator stems from its mathematical simplicity, compared to other oscillators such as the Van der Pol oscillator which has quadratic nonlinearity. In addition, the relation between model parameters and the behaviour of the oscillator can be predicted and hence, its popularity in robotic control.

In this study, an important factor in adopting the Matsuoka oscillator to generate muscle activations was that this oscillator is biologically-inspired. In humans, the neural system generates rhythmic signals that are sent to the musculo-skeletal system in order to produce muscle activations. Since his model is a mathematical representation of neurons or a network of neurons which can generate various types of rhythmic patterns, it can be applied to the modelling of neuronal circuits in the spinal cord. Another important part of his model is its adaptation. When a neuron in the Matsuoka oscillator is excited, its output increases and then slowly decreases to a lower level. This decay is close to what is observed biologically in real neurons (Matsuoka 1985).

Matsuoka oscillators have been previously used to generate net muscle torque at each joint (Taga et al., 1991; Taga 1995; Ishiguro et al., 2003). Parameters needed to produce a smooth torque

that resembles human bipedal locomotion have been estimated arbitrary. This can be achieved by trial and error, since the parameters of the neural oscillators are in proportion to the amplitude produced (Taga 1995). In our study, we wanted to determine if this model with parameters determined by NLS algorithm can, in principle, replicate muscle signals. We do not attempt to explain every intricate detail of the EMG measurements, but to describe the general behaviour of certain muscles. Our study showed that a simple two-neuron model, despite being the simplest mutual inhibition network, can achieve a good fit to experimental EMG data. It should be noted that in some reflex and gait studies, EMG measurements were averaged for each subject or across all trials (Winter 1991; Hof et al., 2002; Kurtzer et al., 2010). This was not done in our study, as using mean values might remove inherent properties in certain muscles (Arsenault et al., 1986). In addition, EMG measurements during gait are usually low-pass filtered at 3-30Hz (Kleissen 1990; Winter 1991; Shiavi et al., 1998). In our study, EMG was filtered at 40Hz so as to preserve the fast transients of the rectified EMG data. Naturally, a better correlation would be achieved if compared to smoother EMG data.

Grillner and Wallen, 1985 suggested that the spinal cord could be made up of different pattern generators responsible for each muscle group in the limbs, and there might be one or several networks made up of a number of neurons that are responsible for locomotion and other movements. However, our study showed that the simplest network of two neurons can explain muscle activation at one joint. Humans can perform multi-joint movements smoothly and yet, they are also able to move one joint of their choice to any desired orientation. So, while a bigger network of neurons might be responsible for an entire walking motion, independent joint motion may require less neurons. In this study, two neurons turned out to be sufficient in explaining muscle activations at the ankle during locomotion. Perhaps, the use of more neurons representing the activation of the gastrocnemius may provide a better explanation as to what is happening at the ankle. The SPG model should then be expanded to include the knee joint since the gastrocnemius is a biarticular muscle.

4.1.2. Parameters

The parameters determined by the NLS algorithm are assumed to be constant throughout the strides (Table 1). It is safe to assume that the SPG can produce constant rhythmic motion and

remains steady during normal unperturbed walking. Naturally, the parameters will change depending on the task required. In our model, the neurons and their interconnections are not elements describing an activity occurring at the cellular level, but instead is a general representation of how internal connectivity in a network of neurons in the spinal cord can be excited by sensory inputs, which in turn, creates locomotion-like muscle activations.

When interconnections or mutual inhibition between the neurons were removed, i.e $a_{12}=a_{21}=0$, oscillations can still occur due to excitatory signal inputs. The control of these parameters, which determine the neuronal properties of the SPG, could possibly come from interneurons, presynaptic inhibition (Matsuoka 1987) or through descending pathways from supraspinal structures. Thus, if these parameter values are low, it may imply that little regulation from the brainstem or intraspinal circuitry is required to generate or modulate rhythmic patterns. From Table 1, b is more than twice the values of a and T. In such a case, the neuron is a "phasic" or "transient-type" neuron (Matsuoka 1985). With two mutually-inhibiting neurons, no oscillations occurred when b=0, regardless of the values of a and T. This meant that adaptation is important for the intrinsic generation of the oscillation in this SPG model. It has been determined that the frequency of the output is correlated to b while inversely correlated to a and T (Matsuoka 1987). We found that the mean fraction of T_2 over T_1 is 0.40±0.50, while b_2 over b_1 is 1.44±0.71. Thus, the SOL has a slower rate of decay and a smaller adaptation effect. Since these parameters affect the rhythmic frequency of the output, the frequency of the TA is higher than the SOL, and this can be demonstrated in Figure 5 where T_2/T_1=0.46, b_2/b_1=1.24.

Parameters m, n, v and w are weights of the excitation to the model (as shown in equations 4-6). These weights affect the amplitude of the outputs, as changing the magnitude of the input s_i will affect the amplitude of the oscillation (Xu et al., 2009). Increasing the weights for only one neuron would increase the amplitude of the output of that particular neuron. Due to mutual inhibition, this led to a decrease in the output of the other neuron. However, this does not always lead to a change in output frequency (Xu et al., 2009). The standard deviations for these parameters were relatively high (Table 1). As a set of parameters was determined for each individual trial, the parameters varied from trial to trial. This meant that each subject would utilise the inputs differently to produce a desired locomotion pattern, though the variation of the

parameters between subjects was not significant (two-way anova, p=0.24). When either one of the two sensory inputs was used, a change of parameters can also produce a locomotor-like output, though the correlation was reduced. This suggested that SPG are capable of adapting to changes and selecting appropriate afferents, so as to enable step-like patterns to occur.

An average of the range between the lower and upper bounds determined at 95% confidence interval was calculated for the parameters for all trials (Table 1). The time lag of the adaptation T had the smallest range. It seemed that the period at which the output is decaying, is instrumental in achieving a good fit. On the other hand, the correlation is less sensitive to changes in *v*, which represents the weight of the changes in hip angles.

4.1.3. Use of inputs

In humans, information about loading may include the mechanoreceptors at the soles of the feet, proprioceptive receptors in the muscles, even the stretch receptors from muscle spindles. However, since it seems that the nervous system utilises information from several load receptors, rather than processing information from each receptor individually (Duysens et al. 2000), the total force calculated from pressure insoles was used as a loading input into the model.

In humans, there are undoubtedly other sensory sources which can influence muscle activation at the ankle. The addition of more inputs might provide an output with a higher correlation. It was found that the contralateral limb has to be able to support the body before ipsilateral swing can begin (Dietz et al. 1994). Naturally, additional information from the contralateral limb, which could determine phase-switching, would enable the SPG to better generate locomotor-like activations. However, a good correlation was achieved by simply using loading and hip angles in this study. This also agreed with previous studies that the two main afferent inputs related to walking in humans is loading and hip position (Dietz and Duysens, 2000).

4.1.4. Supraspinal control

In the present study, we assumed that one is simply walking without any deliberation during steady state walking at self-selected speed. Therefore, a supraspinal input was not included in our model. While the SPG are responsible for basic rhythmic patterns (Ijspeert 2008), some degree

of supraspinal control takes place when a sudden perturbation occurs, or when environmental conditions change. It is still unclear how much control from the brain is required to perform certain corrections to movements (Duysens et al. 2002). Even in patients with incomplete spinal injuries, it is unclear whether supraspinal control is working together with SPG or completely in command during locomotion (Duysens et al. 1998; Pang and Yang 2000). However, Geyer and Herr, 2010 demonstrated that it might also be possible that spinal reflexes can dominate over a central input in controlling locomotion. Our study demonstrated that a supraspinal control is not required to generate realistic muscle activations at the ankle during normal steady-state walking.

4.2. Case 1 – Sudden loss in loading afferents

Co-contraction of the SOL and TA was found in our model when perturbation was introduced during stance. It was suggested that this co-activation might be the first defensive response to sudden changes in a support surface to increase joint stability (Misiaszek et al., 2000; Nakazawa et al., 2004; Shinya et al., 2009).

The SOL results from our model without a supraspinal input were similar to the perturbation trials (Table 2). So, it would seem that the neural network at the spinal level could be sufficiently capable in responding to perturbations without interference from higher centres. Nakajima et al., 2000 had suggested that the propriospinal neural network is less likely to play a major role in human movements when a strong corticospinal neural network exists. However, in our view, an appropriate response might be performed even at the lowest basic level since maintaining dynamic stability on the stance limb is more crucial in bipedal than quadruped walking. The increase in SOL activity was found to be significant only during the loading response of gait. It has been emphasized that the most important period during stance phase is soon after heel strike when the body weight is being shifted from one limb to another (Christensen et al., 2000). Since this phase requires the ipsilateral limb to quickly access information on whether it can support the person's weight, an absence of an expected loading afferent would activate the SOL to act as a braking mechanism (van der Linden et al., 2007) and/or to quickly achieve joint stability (Nakazawa et al., 2004).

For perturbation at mid- to late stance, the TA showed a significant increase during unloading in our model (Table 3). An increase in TA activity will be expected when plantar afferents fail to receive a loading input. This is because the spinal cord might perceive a swing phase, and thus activate the TA for toe clearance. However, our perturbation trials performed on healthy subjects only showed significant differences during midstance. The different results could be due to the presence of other sensory afferents in an intact human. With other available afferent sources, the effect of a single sensory input like loading is reduced. In addition, the availability of a descending pathway could be utilised. The TA is thought to be under greater supraspinal control particularly prior to phase change (Schubert et al., 1997; Field-Fote and Dietz, 2007). Nevertheless, it remains uncertain here if the TA receives more modulation from supraspinal or other afferent sources during sudden unloading.

Previous experimental studies on human subjects have examined the effects of a drop in ground support and reported different results. Increase in activity of the ankle extensors and flexors were found in Nakazawa et al., 2004, Marigold and Patla, 2005 and van der Linden et al., 2007 while af Klint et al., 2009 reported a decrease in SOL activity. While there were differences in the phases at which loss of ground support was applied, we think the main difference could be due to the drop height. A study by af Klint et al., 2009 caused a platform to accelerate downwards by 8 cm, while other studies were between 1 cm (Nakazawa et al., 2004) to 6.5 cm (Shinya et al., 2009). af Klint et al., 2009 had not reported on hip angles in their study but we postulate that a larger drop might produce significant changes in hip position. A delayed advancement of the hip over the foot might result in a continuing SOL activity. In our model, we retained similar hip joint angles during the perturbation just as in normal walking. A study by Shinya and Oda, 2010 had their subjects walked into a hidden hole of 8.5 cm, but they did not report on the resulting increase or decrease in the SOL or TA.

A sudden perturbation to a limb will result in complex reflex responses. So, the question of whether the muscle activities are of short or long latency responses arises. Shinya et al., 2009 found increases in SOL and TA activities with latencies of 117ms and 126 ms respectively, when a sudden loss of ground support occurred at early stance. Nakazawa et al., 2004 and Marigold and Patla, 2005 also reported long-latency muscle responses (~140-160ms) due to changes in

support surfaces. Nieuwenhuijzen et al., 2002 found long-latency muscle responses (>100ms) that could stabilize the ankle during landing in an inverted platform. So, it would seem that results from our model are mostly in agreement with what occurred at longer latencies. Now, the following question would be whether the responses are influenced by spinal or supraspinal sources. Shinya and Oda, 2010 reported that response latencies can be affected by prior knowledge about a potential perturbation. They reported a faster SOL response from expected conditions than unexpected conditions. So, supraspinal structures including the cerebellum could play a role in the responses to a change in ground support. While it is generally agreed that the first component of the spinal stretch reflex is due to monosynaptic activation involving the Ia afferents, the supraspinal origins of the second and third components in an elicited EMG activity are still being questioned. While it is usually assumed that long latencies are attributed to higher centres, other studies have supported spinal pathways. Eklund et al., 1982 reported that the motor bursts are due to muscle oscillations as a result of movement. A supraspinal contribution to the muscle activity in the perturbation trials performed on healthy subjects cannot be ruled out, though our SPG model showed an increase in muscular activity in the absence of a supraspinal input (Tables 2-3). Nevertheless, in our study, the response of the SOL was similar in both our model and perturbation trials. So, responses to load-related perturbations in humans can still be modulated at the spinal level (Dietz and Duysens, 2000). However, this was only seen in the perturbed trials during midstance for the TA. So, modulation of the TA at loading response and late stance needs more clarification.

While our SPG model with two neurons and two afferent inputs successfully yielded muscle activations that closely fit EMG data during normal walking (Figures 5-6), our approach with using the model for perturbed walking might be simplistic; SPG may utilise several sensory afferents to generate muscle activity (Dietz and Duysens, 2000), perhaps even with overlapping effects. Another limitation is the 16 parameters used. A larger number of parameters would produce a better fit to muscle activations. In addition, the model also did not take the contralateral limb into consideration. During perturbation, a rapid ipsilateral response is evoked for re-positioning of the foot, while a rapid contralateral response is evoked to achieve equilibrium (Berger et al., 1984; van Dieën et al., 2007).

4.3. Case 2 – Change in walking speed

While significant differences were found in both inputs (loading and hip angles) for the different walking speeds and silly walks (Table 4), no significant differences were found in the model parameters for normal walking in all speeds (Figure 14). For normal walking, this might imply that an insignificant change in a parameter is sufficient to cause a significant change in the output. Since the control of these parameters, which determine the neuronal properties of the SPG, could possibly come from interneurons, presynaptic inhibition (Matsuoka 1987) or through descending pathways from supraspinal structures, the insignificant changes might imply that regulation from the brainstem or inter-spinal circuitry is not required to modulate the activation patterns during walking. So, while a cortical input is required for the initiation and stopping of a motion, higher command centres or an additional group of neurons need not be recruited to regulate motor output during steady-state walking regardless of speed. It has been suggested that the motor control mechanism responsible for gait transitions between walking and running could be attributed to the same neural circuitry (Labini et al., 2011). Our study supports their idea that the same neural network could be responsible for different types of gait.

Grasso et al., 1998 suggested that the nervous system attempts to meet motor demands by controlling posture or limb joint motion, rather than regulate muscle activations. We agree with their arguments, as we had successfully used loading and hip angles as inputs to the SPG model to generate muscle activations. In addition, so long as posture is not disturbed, the same neural network will be utilised (Lamb and Yang, 2000). Since the data was captured during steady-state walking, it might also be important that the alternating activations between the flexor and extensor are not disrupted. Perhaps, changes to gait components are secondary, and might be a result of changes to step length, rather than the result of a different motor control mechanism.

It has been shown that cats with lesions in the motor cortex have no problems walking on a flat horizontal surface, till they were required to cross over obstacles or go up a ladder (Armstrong 1988). In addition, Armstrong and Drew 1984 found that pulses measured in the cortical neurons of cats were unrelated to speed, though significant increase was found in muscle activity. So likewise in humans, conscious effort is not necessary during level walking regardless of the pace

at which it was performed, until we come across an obstacle or a sudden change to our external environment where corrective responses are required.

4.4. Case 3 – Silly walks

While significant differences were found in both inputs (loading and hip angles) between normal walking and silly walks (Table 4), significant differences were only found in parameter T_2 (Figure 14). We were expecting more differences in the silly walks since the muscle activations are due to acting on a command from the brain to perform something silly. However, we found significant changes only for T_2 which is the adaptation time constant for the TA. It is known that persistent inward currents (PIC) levels play an essential role in the firing activities of motoneurons (Li et al., 2004). It was speculated that PIC are expressed in the extensors from birth, but less so in the flexors (Cotel et al., 2009). This is because while the extensors are mostly activated during walking, the flexors do not require long lasting bursts. So it would be more functional to modulate the flexors, rather than the extensors. Nevertheless, it remains uncertain here if the TA requires more intervention from the brainstem or additional neural circuitries than the SOL. The significant difference in T_2 could also be due to the SPG model, which requires a strong adaptation effect in generating stable oscillations (Matsuoka 1985; $T_{2,\text{ silly walks}}$=0.12±0.17 compared to $T_{2,\text{ self-selected}}$=0.03±0.04). So, the higher value in T_2 could just be a way for the model to continue generating oscillations.

A limitation in this study is the limited array of silly walks the subjects can perform while walking on a treadmill at a constant speed. In addition, since the data was captured on a treadmill, the movements they performed still involved a continuous alternating activations between the antagonistic muscles at the ankle. So since the rhythmic patterns activations between the flexor and extensor were not disrupted, the same motor control mechanism from the SPG was utilised. If the subjects were given a freer hand on the type of silly walks they would like to perform, just like those seen in The Ministry of Silly Walks, significant differences in more model parameters might be found.

5. Conclusion

In conclusion, a simple two-neuron SPG model was able to yield muscle activations at the ankle. It is possible that afferent inputs from loading and hip orientation are largely under the control of SPG, which in turn, may influence the muscle activations. A close correlation between simulated and measured muscle activations also indicates that spinal control should not be underestimated in models studying human locomotion.

We found that SPG in the spinal cord can interpret and respond accordingly to velocity-dependent afferent information. Changes in walking speed do not require a different motor control mechanism so long as equilibrium is not affected, and there is no disruption to the continuous rhythmic patterns produced at the ankle. In addition, our model showed co-activation of antagonistic muscles when sudden unloading was introduced during stance. This could be the first defensive response to sudden changes in a support surface to increase joint stability. For the SOL, simulated EMG data from our model was similar to experimental perturbed trials. However in the TA, this was only found during midstance. So, less than expected afferents to the spinal cord might require mediation from other sources for the TA. Furthermore, only the adaptation time constant for the TA was significantly different while performing silly walks compared to normal walking. So, further studies will have to investigate if the TA requires more modulation from supraspinal or other afferent sources during perturbed walking or while performing silly walks.

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